Hemorrhage into the Brain Abscess Cavity with Fallot’s Tetralogy

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1016–2291/05/0000–0000 $22.00/0
Accessible online at: www.karger.com/pne

Key Words
Brain abscess · Fallot’s tetralogy · Hemorrhage

Abstract
Hemorrhage into the abscess cavity is a complication of brain abscess. It has been reported to be due to inflammation which results in the damage of the fragile neovasculature of the abscess wall. Hypoxia caused by Fallot’s tetralogy or other congenital heart diseases facilitates the damage of these vessels with the lacking supportive tissues, and in turn intracavital bleeding.

A 6-year-old male patient who was diagnosed as having Fallot’s tetralogy at 5 months of age suddenly developed unconsciousness while being observed in the pediatric department due to respiratory distress. On examination, the Glasgow Coma Score was found to be 13. The patient had intermittent fever with blood O2 saturation level at 87, white blood cell count 12,400/mm3, hemoglobin level 19 g/dl, and hematocrit level 63%. A brain abscess with an intracavital hemorrhage was delineated on the right temporal region of the computed tomography (fig. 1).

Purulent bloody discharge was drained from the burr hole during the operation. Since the culture had no pathogenic microorganism growth, broad-spectrum antibiotic (cephalosporin, aminoglycoside and vancomycin) treatment was administered for 8 weeks, at the end of which the patient’s cerebral abscess had completely resolved.

Fig. 1. A brain abscess with intracavital hemorrhage (arrow) and smooth encircling ring enhancement is delineated in the right temporal lobe on contrast axial CT.
Discussion

In cases with brain abscess, the incidence of congenital heart disease varies from 3.4 to 13.5% [1, 2]. This is more likely to happen in congenital heart diseases in which pulmonary hypertension predominates or the right left shunt exacerbates [1]. Histological investigation of the abscess showed a newly developed vasculature in the collagenous capsule surrounding the necrotic core which was fragile due to release of angiogenic factors from macrophages and other inflammatory cells [3]. Although the physiopathology of the intra-abscess hemorrhage is not well understood, intense inflammation has been reported to be the possible cause of damage to the fragile vasculature [3]. Hypoxia, by increasing the damage to the vessels with weakened supportive tissue, is known to cause the bleeding process [4]. In our present case with congenital heart disease, the severe increase in hematocrit level as well as the hypoxia due to the right left shunt, which hampered the thin vascular circulation, increased the ischemic event which already existed. We believe that the free radicals due to ischemia probably facilitated the vulnerability of these neovascular tissues.

References